bupivacaine, however, is a "fast-in, slow-out" agent. As a result of the slow recovery with the use of bupivacaine, a substantial frequency-dependent block accumulates at heart rates between 60 and 150 beats per minute (slow recovery from block during diastole). Bupivacaine is therefore more cardiotoxic than lidocaine at clinically equivalent local anesthetic concentrations; bupivacaine is potentially cardiotoxic when a large dose (probably 1 mg per kg of body weight or greater) is given intravascularly.

In August 1984, with an increasing number of reported deaths related to accidental intravascular bupivacaine injection, mostly among the obstetric population, the Food and Drug Administration issued urgent new recommendations about bupivacaine, stating that the 0.75% concentration is no longer recommended for obstetric anesthesia. The reason for the increased incidence of cardiotoxic reactions to bupivacaine in pregnancy is not clear. It may be due to the more frequent use of bupivacaine for obstetric epidural blocks or possibly that the physiologic changes during pregnancy make a woman more susceptible to such reactions, or more difficult to resuscitate, than a nonpregnant woman.

Despite its potential cardiotoxicity, bupivacaine remains a very useful agent for regional anesthesia, with a longer duration of action than lidocaine and, in lower concentrations, the ability to produce a high-quality analgesic block with minimal motor block. With careful administration and using meticulous techniques of test dosing and slow administration of a dose in fractional amounts, bupivacaine can be used effectively and safely. It is apparent, however, that great caution must be exercised to prevent an accidental intravascular injection of a large dose of bupivacaine, whatever the concentration.

MARK A. ROSEN, MD
San Francisco

REFERENCES

Albright GA: Cardiac arrest following regional anesthesia with etidocaine or bupivacaine (Editorial). Anesthesiology 1979 Oct; 51:285-287

Clarkson CW, Hondeghem LM: Mechanism for bupivacaine depression of cardiac conduction: Fast block of sodium channels during the action potential with slow recovery from block during diastole. Anesthesiology 1985 Apr; 62:396-405

Kotelko DM, Shnider SM, Dailey PA, et al: Bupivacaine-induced cardiac arrhythmias in sheep. Anesthesiology 1984 Jan; 60:10-18

Rosen MA, Thigpen JW, Shnider SM, et al: Bupivacaine-induced cardiotoxicity in hypoxic and acidotic sheep. Anesth Analg 1985; 64:1089-1096

Hypokalemia and Potassium Administration in the Perioperative Period

PERIOPERATIVE HYPOKALEMIA has given anesthesiologists cause for concern due to the accepted relationship between low serum potassium levels and cardiac dysrhythmias. Two practices resulting from this concern have recently been questioned: automatic postponement of elective surgical procedures when a serum potassium level is below arbitrarily chosen levels, resulting in inconvenience to the patient and the surgical team and a pronounced increase in the cost of hospital care; second, aggressive intravenous replacement of potassium that in itself can precipitate serious morbidity and mortality.

Serum potassium levels reveal little information about the total body exchangeable potassium (representing 0.4% of the total body potassium). If a serum potassium concentration of 4.0 mEq per liter is considered normal, a potassium level of 3.0 mEq per liter reflects a 25% deficit of total body potassium. In a 70-kg adult, this represents 1,100 mEq of potassium, too great a deficit to replace rapidly with safety. Acute hypokalemia can be seen during anesthesia; for example, a decrease in a serum potassium level of 1 mEq per liter can result from hyperventilation that reduces the arterial carbon dioxide pressure from 45 torr to 25 torr, resulting in no loss of total body potassium and a 12-mEq transfer from the extracellular to the intracellular compartment of potassium ions. Replacement in this situation is unnecessary and carries the risk of a dangerously high serum potassium level being attained.

Studies by Vitez and colleagues of patients with chronic hypokalemia and by Allard and Cheek of patients with acute hypokalemia seen intraoperativelay both suggest that the dangers of hypokalemia in the perioperative period may have been overstated and may be less than that of iatrogenic hyperkalemia from overadministration or too rapid an administration of potassium.

The commonly accepted potassium levels for an elective operation (3.0 mEq per liter for chronic hypokalemia and 3.5 mEq per liter for hypokalemia and digitalis therapy) are arbitrary generalizations that the study of Vitez and associates would suggest are too high to routinely cancel surgical procedures. The interpretation of a single potassium level should be judged individually in light of the clinical situation within which it is found, and electrocardiographic evidence of hypokalemia should be elicited. If potassium is to be administered in a patient with chronic hypokalemia, it should ideally be given before the admission of the patient for an operation. If hypokalemic-related cardiac dysrhythmias occur and intravenous repletion of potassium is considered necessary, it should be administered in dilute solution through a central line at a maximum rate of 0.5 mEq per kg per hour with continuous electrocardiographic monitoring.

MARTIN W. ALLARD, MD
Loma Linda, California

REFERENCES

Allard MW, Cheek DBC: Omission of supplementary potassium perioperatively in cardiac surgery (Abstr). Anesth Analg 1985 Feb; 64:189

McGovern B: Hypokalemia and cardiac arrhythmias. Anesthesiology 1985 Aug; 63:127-129

Vitez TS, Soper LE, Wong KC, et al: Chronic hypokalemia and intraoperative dysrhythmias. Anesthesiology 1985; 63:133-135

Malignant Hyperthermia Update

IN THE EARLY 1970s, malignant hyperthermia was the leading cause of anesthetic deaths, with an estimated mortality rate of 65% to 80%. By the mid-1980s mortality rates have dropped to below 5%. This dramatic change in statistics can be attributed to several factors: an awareness by the medical and lay community, with the increased detection of persons who might be susceptible to malignant hyperthermia; the early detection and treatment of malignant hyperthermia by anesthesiologists, and the availability (1979) of injectable dantrolene sodium, a drug that effectively reverses a malignant hyperthermia crisis.

Typically the syndrome is manifested by sinus tachycardia, a rising blood pressure and tachypnea. The skin becomes mottled—cyanotic with patches of bright red flushing. Rigor mortis-like stiffening of masseter or all skeletal muscles may develop. The temperature increase that results from the hypermetabolic condition in skeletal muscle occurs relatively